# Dizziness and Vertigo

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**KEYWORDS**

• Benign paroxysmal peripheral vertigo • Vestibular neuritis • Vestibular migraine
• Meniere's disease • Migrainous vertigo • Acute labyrinthitis

**KEY POINTS**

- Benign paroxysmal peripheral vertigo (BPPV) is the most common cause of vertigo. It is diagnosed using the Dix-Hallpike maneuver and treated with the Epley maneuver.
- Vestibular neuritis is a single episode of acute, severe vertigo. The head thrust test and visual fixation can help differentiate it from acute stroke. The mainstay of treatment is vestibular rehabilitation.
- Vestibular migraine manifests as vertigo accompanied by classic migraine symptoms, and responds to migraine medications.
- Over eighty percent of patients with Meniere's disease can be successfully managed with lifestyle changes and diuretics.

## INTRODUCTION

Dizziness is a common and challenging condition seen in the primary care office. More than one-third of Americans see a health care provider for dizziness during their lifetime.[1] Although most dizziness is due to benign causes, life-threatening causes, such as a stroke or intracranial mass, also need to be excluded. Because "dizziness" is a vague term that can include a wide array of medical disorders, it is important to use a stepwise approach to differentiate between causes.

First, clinicians should distinguish between the four common types of dizziness: (1) presyncope, (2) disequilibrium, (3) psychogenic dizziness, and (4) vertigo. Patients should be asked to specifically describe their dizziness in their own words. Vertigo is a false sense of motion of either the environment or self. Often, patients describe a feeling of the room spinning or tilting. Benign paroxysmal peripheral vertigo (BPPV), vestibular neuritis, vestibular migraine, and Meniere's disease are the four most common causes of vertigo in ambulatory settings, and a thorough history and physical examination alone can lead to the diagnosis in most cases (**Table 1**).

## BENIGN PAROXYSMAL PERIPHERAL VERTIGO

BPPV is the most common cause of vertigo. Patients typically report brief episodes triggered by head movement. A positive Dix-Hallpike maneuver is diagnostic, and

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**Table 1**
**Characteristics of common causes of vertigo**

| | BPPV | Vestibular Neuritis | Vestibular Migraine | Meniere's Disease |
|---|---|---|---|---|
| Time Course | Recurrent, lasting seconds | Single episode lasting days | Recurrent, lasting minutes to days | Recurrent, lasting hours |
| History | Brief, triggered by head movement | Subacute onset of severe, constant vertigo with significant nausea and vomiting | Previous history of migraine. Vertigo accompanied by migraine symptoms | Hearing loss, tinnitus and ear fullness |
| Nystagmus | Up-beating torsional | Horizontal or horizontal-torsional | Usually none | Horizontal or horizontal-torsional |
| Gait | Normal | Veers toward affected side | Abnormal during vertigo attacks | May have impaired gait and imbalance |
| Auditory Symptoms | None | Hearing loss (acute labyrinthitis) | None | Present |
| Diagnostic Findings | Positive Dix-Hallpike maneuver | Positive head-thrust test, Nystagmus suppressed by visual fixation | Vertigo attacks resolve with acute migraine medications | Repeat audiometry shows fluctuating, low-frequency hearing loss |

canalith repositioning procedures (CRP), such as the Epley maneuver, are the mainstay of treatment.

### *Epidemiology*

BPPV accounts for more than 40% of vertigo diagnoses seen in primary care, and is the most common cause of vertigo across the lifespan.[2] Patients with BPPV most commonly present between the fifth and seventh decades of life, and it is seen more commonly in women.[3] By 80 years of age, nearly 10% of adults have been diagnosed with BPPV during their lifetime.[4]

### *Risk Factors*

A history of prior head trauma or prior vestibular disorders, such as vestibular neuritis, increases a patient's risk of BPPV. Osteoporosis and vitamin D deficiency have been associated with BPPV.[5] Recently, sleep position has also been correlated with BPPV, with patients who have BPPV being more likely to report lying on their sides with the affected ear down.[6]

### *Pathophysiology*

It is hypothesized that BPPV is caused by loose calcium carbonate debris (otoconia) in the semicircular canals of the inner ear. With head motion, otoconia begin to move freely in the canals. When head motion stops, otoconia continue to move, causing endolymph to move against the hair cells of the semicircular canal. This leads to a false sense of motion that lasts until the otoconia settle, usually only a few seconds. The posterior canal is involved in 85% of cases, followed by the horizontal canal in 10% of cases.[7] Rarely, BPPV can be bilateral.

### *Clinical Presentation*

In BPPV, patients complain of brief episodes of vertigo triggered by position changes. Episodes usually last only seconds, and are less than 1 minute in duration. Commonly, patients experience attacks when rolling over in bed, or tilting the head to look upward. Patients may report difficulty placing objects on high shelves, or bending forward to tie shoes. Nausea and vomiting may occur with episodes. Vertigo from most causes is exacerbated by certain movements. In BPPV, however, vertigo is actually *preceded* by position changes and patients are normal between attacks.

Physical examination should include a complete ear, nose, and throat, cardiovascular, and neurologic evaluation. This is to exclude other causes of vertigo (**Table 2**), because there are no specific physical examination findings in BPPV. However, the Dix- Hallpike and supine roll tests are two maneuvers that can confirm BPPV suspected by historical clues.

### *Diagnosis*

The diagnosis of BPPV is made clinically. It is confirmed most often with the Dix-Hallpike maneuver, and in some cases the supine roll test.

#### *Diagnostic maneuvers*

The Dix-Hallpike maneuver should be performed in any patient being evaluated for BPPV (**Fig. 1**). It is used to diagnose posterior canal BPPV. Before performing the Dix-Hallpike, clinicians should warn patients that severe vertigo, and possibly nausea, may occur. Patients sit upright on an examination table with the head rotated 45 degrees to the right. Maintaining this head position, the examiner quickly lays the patient back into the supine position, and extends the neck approximately 20 degrees so that the head "hangs" supported off the edge of the examination table. The examiner then observes the patient for vertigo and nystagmus. In posterior canal BPPV, nystagmus is up-beating and torsional. Characteristically, there is a latency period of 5 to 20 seconds after the position change to the onset of nystagmus and vertigo. The nystagmus and vertigo initially increase in intensity and then resolve within 60 seconds ("crescendo-decrescendo" nystagmus). The side that provokes symptoms indicates the involved ear. Even if positive, the Dix-Hallpike should be repeated on the opposite side to exclude bilateral BPPV. A positive Dix-Hallpike test requires observation of the characteristic nystagmus. If a persistent or down-beating nystagmus is elicited, a central cause should be suspected.

If the Dix-Hallpike maneuver is negative, a supine roll test should be performed to diagnose lateral canal BPPV (**Fig. 2**). For the supine roll test, the patient begins in the

**Table 2**
**Differential diagnosis of vertigo**

| Peripheral Causes | Central Causes |
|---|---|
| BPPV | Migrainous vertigo |
| Vestibular neuritis | Intracranial mass |
| Meniere's disease | Cerebrovascular attack |
| Perilymphatic fistula | Vertebrobasilar insufficiency |
| Herpes zoster oticus | Chiari malformation |
| Acoustic neuroma | Multiple sclerosis |
| Ototoxicity | Episodic ataxia type 2 |
| Otitis media | |
| Semicircular canal dehiscence syndrome | |
| Posttraumatic vertigo (labyrinth concussion) | |

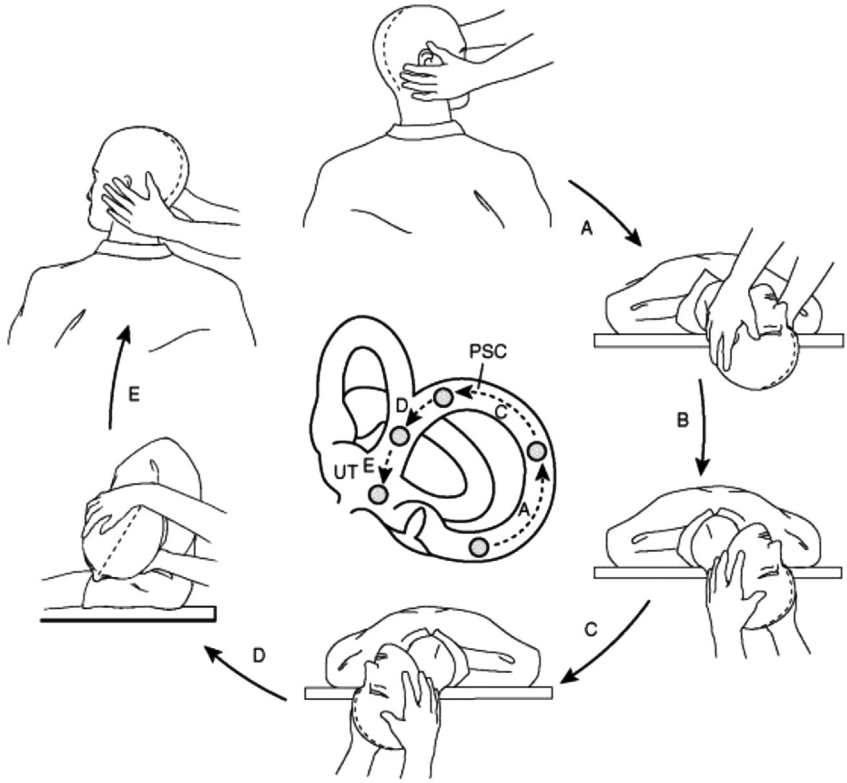


**Fig. 1.** Treatment maneuver for benign paroxysmal positional vertigo affecting the right ear. To treat the left ear, the procedure is reversed. The drawing of the labyrinth in the center shows the position of the particle as it moves around the posterior semicircular canal (PSC) and into the utricle (UT). The patient is seated upright, with head facing the examiner, who is standing on the right. (A) The patient is rapidly moved to head-hanging right position (Dix-Hallpike test). This position is maintained until the nystagmus ceases. (B) The examiner moves to the head of the table, repositioning hands as shown. (C) The head is rotated quickly to the left with right ear upward. This position is maintained for 30 seconds. (D) The patient rolls onto the left side while the examiner rapidly rotates the head leftward until the nose is directed toward the floor. This position is then held for 30 seconds. (E) The patient is rapidly lifted into the sitting position, now facing left. The entire sequence should be repeated until no nystagmus can be elicited. After the maneuver, the patient is instructed to avoid head-hanging positions to prevent the particles from reentering the posterior canal. (*From* Rakel RE. Conn's current therapy 1995. Philadelphia: WB Saunders; 1995. p. 839; with permission.)

supine position with their head facing upward. The examiner quickly moves the patient's head 90 degrees to one side, and examines for nystagmus and vertigo. The supine roll test should be repeated to the opposite side. In lateral canal BPPV, nystagmus is most often a geotrophic type: horizontal and beating toward the lower (affected) ear during the supine roll test. When rolled to the opposite side, the nystagmus recurs and beats toward the lower (unaffected) ear but is less intense. Less often, nystagmus may be of apogeotrophic type: horizontal and beating toward the upper ear.

If the Dix-Hallpike and supine roll tests are negative, another diagnosis should be suspected. However, if the clinical history is strongly indicative of BPPV, then the

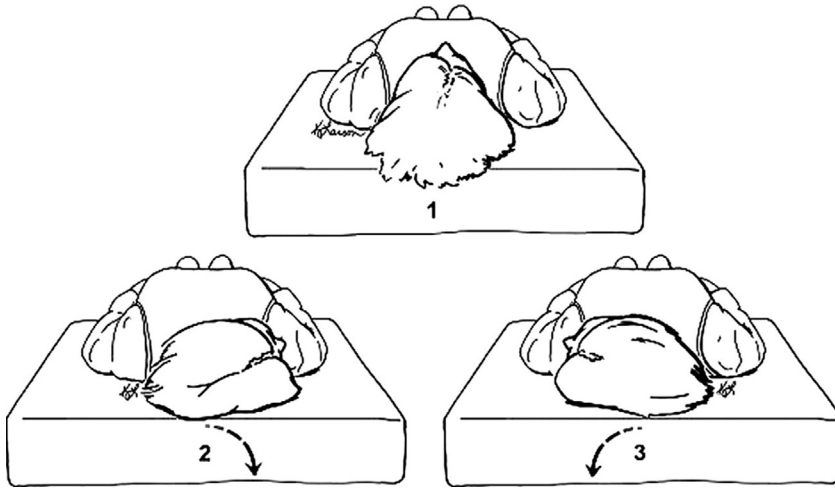


**Fig. 2.** Supine roll test. (1) Start supine with head in neutral position. (2) Turn the patient's head quickly 90 degrees to the right and observe for nystagmus. Return the head to neutral position (1). (3) After any residual nystagmus or symptoms resolve, turn the patient's head quickly 90 degrees to the left and observe for nystagmus. (*From* Fife TD, Iverson DJ, Lempert T, et al. Practice parameter: therapies for benign paroxysmal positional vertigo (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2008;70:2067–74; with permission.)

patient should return in 1 week to repeat the maneuvers. Although the positive predictive value of the Dix-Hallpike maneuver is 83%, its negative predictive value is 52%.[2] Therefore, it is worth repeating to avoid a false-negative result.

***Radiographic imaging***

Patients with BPPV do not require neuroimaging unless the diagnosis is uncertain and a central cause is suspected (**Table 3**). Magnetic resonance imaging (MRI) is the best imaging test in most cases because it includes the posterior fossa, and is useful for evaluating central causes, such as a cerebrovascular lesion, intracranial mass, or demyelinating disease.

**Table 3**
**Red flags for a central cause**

| |
|---|
| History |
| Sudden onset |
| New, severe headache |
| Cardiovascular risk factors |
| Nystagmus |
| Direction-changing |
| Purely vertical or torsional |
| Unsuppressed by visual fixation |
| Inability to walk |
| Negative head-thrust test |
| Additional neurologic signs (e.g. aphasia, dysarthria, weakness, sensory loss) |

### *Vestibular function testing*

Vestibular function testing is indicated when the diagnosis is uncertain or BPPV is resistant to treatment. Assessment involves an array of specialized tests that record nystagmus in response to caloric stimulation, position change, and voluntary eye movements. Vestibular function testing may also provide prognostic information for treatment planning in severe cases.

## *Treatment*

Treatment options for BPPV include CRP, vestibular rehabilitation, observation, and surgery. Recurrence of BPPV is common, with at least 25% of patients experiencing a repeat episode within 6 months.[8] Recurrence is more likely in older patients and those with prior head trauma.

### *Epley maneuver*

The Epley maneuver is a highly effective, safe, canalith repositioning treatment of posterior canal BPPV that can be offered in the office setting (see **Fig. 1**).[7] This procedure consists of a series of head position changes that essentially settle the loose otoconia from the semicircular canal into the utricle, where they can no longer trigger vertigo attacks. The Epley maneuver is effective in more than 90% of cases, and has an odds ratio of 4.2 (95% confidence interval, 2.0–9.1) for symptom resolution.[9] Patients who suffered severe nausea or vomiting with the Dix-Hallpike may require pretreatment with an antiemetic. The liberatory (Semont) maneuver can be tried for patients who cannot physically undergo the Epley maneuver. Postural restrictions, such maintaining upright posture and limiting cervical motion, may add a slight benefit to CRP alone.[10] Home CRP exercises are also effective, and may be especially useful for expedient self-treatment if future episodes recur.[11]

### *Lempert maneuver*

The Lempert maneuver (barbeque roll maneuver) treats lateral canal BPPV.[12] Lying supine, the patient is rolled 90 degrees and held until symptoms stop. This is continued for a full 360 degrees. Interestingly, a case report also noted successful treatment of lateral canal BPPV with repeated somersaults.[13]

### *Vestibular rehabilitation*

Vestibular rehabilitation is a series of physical therapy exercises that improve central compensation for a peripheral deficit causing vertigo. Exercises usually consist of moving the head while eyes are fixed on an object, or moving the body while moving the head. Although less effective than CRP in the short-term, vestibular exercises are more effective than observation, and reach similar effectiveness rates to CRP by 3 months' follow-up.[14] Furthermore, vestibular rehabilitation may prevent recurrence of BPPV, especially in the elderly.

### *Pharmacologic*

Treatment with vestibular-suppressant medications, such as benzodiazepines and antihistamines, should be avoided in BPPV. These medications can blunt central compensation, and increase risk of falls. Furthermore, there is no evidence that treatment of BPPV with vestibular-suppressant medications is effective.[7]

### *Observation*

Because BPPV can remit spontaneously, usually over the course of 4 to 6 weeks, observation is a potential treatment option. Observation may be considered for patients with mild BPPV, or those who may not tolerate CRP or vestibular rehabilitation.

However, patients are at higher risk of falls until BPPV resolves and recurrence is more likely.

#### *Surgical treatment*

Surgery is rarely indicated, and is reserved for severe, refractory cases of BPPV. Surgically occluding the posterior canal with a plug is effective in 90% of cases, with 5% experiencing permanent hearing loss.[15] Other surgical options include laser ossification of the posterior canal and posterior ampullary nerve transection.

## VESTIBULAR NEURITIS

Vestibular neuritis is an acute, prolonged attack of severe vertigo that is thought to be of viral origin. Symptomatic care is the mainstay of treatment, because most cases resolve spontaneously with complete recovery. Although a benign disorder, it must be differentiated from more serious causes of acute vertigo, such as a cerebrovascular accident.

### *Epidemiology*

Vestibular neuritis is the second most common cause of vertigo. It accounts for almost 10% of all patients seen for dizziness.[16] Most patients diagnosed are 30 to 50 years of age. Men and women are affected equally. There are no well-studied risk factors for vestibular neuritis.

### *Pathophysiology*

Vestibular neuritis is thought to be caused by a viral infection of the eighth cranial nerve. Supporting evidence includes an increased incidence of vestibular neuritis during viral epidemics, and the common precedence of vestibular neuritis by a viral syndrome. Herpes simplex virus may be an etiologic agent, because studies have identified herpes simplex virus-1 DNA in the vestibular ganglia of patients with vestibular neuritis.[17] Viral infection may lead to inflammation, and potentially atrophy, of the vestibular nerve causing severe vertigo.

### *Clinical Presentation*

Vertigo is sudden in onset, persistent, and severe. Patients may report awakening with severe vertigo, or may experience a subacute onset, with vertigo worsening over several hours. Vertigo is most severe for the first 1 to 2 days, and then gradually improves over several weeks. Initially, patients may have significant nausea and vomiting. Any motion worsens the vertigo; therefore, many may prefer to lie still with their eyes closed.

On physical examination, patients have a spontaneous nystagmus at the onset of illness. The nystagmus is unidirectional and horizontal/horizontal-torsional, with the fast phase beating away from the affected side. The nystagmus can be suppressed by visual fixation. Visual fixation can be tested by asking the patient to focus on an object in the room (nystagmus ceases) and then placing a blank sheet of paper in front of the patient's face (nystagmus resumes). Importantly, a central lesion, such as acute stroke, often presents with spontaneous nystagmus unsuppressed by visual fixation.

When assessing gait, patients with vestibular neuritis tend to veer toward the affected side. An inability to walk, however, is a red flag for a central cause. Hearing is normal in vestibular neuritis. When hearing loss is associated, the condition is known as acute labyrinthitis. Other neurologic signs and symptoms are not found in vestibular neuritis; their presence should raise concern for a central cause.

### *Diagnosis*

Like BPPV, the diagnosis of vestibular neuritis is made clinically. Specialized physical examination maneuvers, such as the head-thrust test, can help differentiate between vestibular neuritis and a more concerning central lesion, such as acute stroke.

#### *Head-thrust test*

The head-thrust test is a useful maneuver to differentiate vestibular neuritis from a central cause (Fig. 3). The head-thrust test is performed by quickly moving the patient's head 10 degrees to the right and left while the patient's eyes remain fixed on the examiner's nose. If a saccade (patient's eyes move briefly off target) is present, then the test is positive for a peripheral lesion. Central vertigo does not exhibit a saccade.

#### *HINTS: three steps to rule out stroke*

The head-impulse-nystagmus-test-of-skew (HINTS) is a combination of three clinical signs that can be tested in patients with acute vertigo to differentiate between

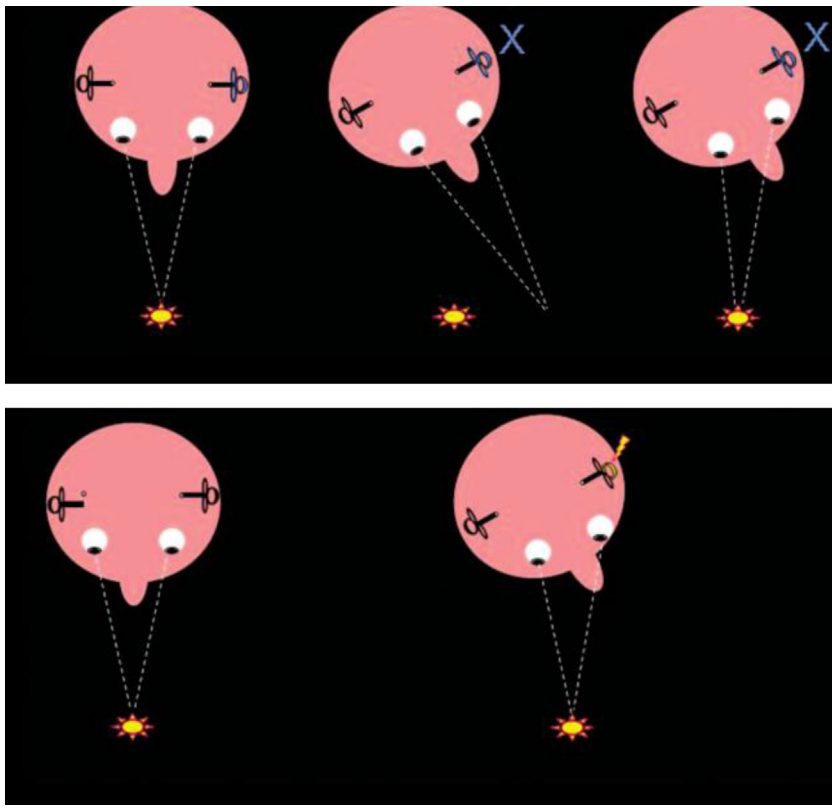

**Fig. 3.** Head-thrust test. *Top panel* shows a positive head-thrust test. The examiner moves the patient's head quickly 10 degrees to the side, in this case to the patient's left. A catch-up saccade is observed when the patient looks away and then refixes on the visual target, indicating a peripheral lesion on the left. *Bottom panel* shows a normal head-thrust test. The patient maintains visual fixation during head movement. (*From* Seemungal BM, Bronstein AM. A practical approach to acute vertigo. Pract Neurol 2008;8:211–21; with permission.)

vestibular neuritis and stroke. These signs include the head-thrust test, nystagmus, and skew deviation. Nystagmus that is bidirectional, purely vertical, or purely torsional is concerning for stroke. Skew deviation (supranuclear vertical eye misalignment, or vertical strabismus) is assessed with the cover/uncover test. Any patient with one or more of these signs should be evaluated for stroke. Using HINTS, stroke can be detected with 100% sensitivity and 96% sensitivity.[18]

#### *Radiographic imaging*

Neuroimaging, such as MRI, is reserved for patients with additional risk factors for stroke, additional neurologic signs, severe headache, or as needed to rule out a suspected central lesion based on clinical findings.

#### *Vestibular function testing*

Vestibular testing may be useful if the diagnosis is unclear. In vestibular neuritis, testing demonstrates a unilateral vestibular loss.

### *Treatment*

Symptomatic care and vestibular rehabilitation are the mainstays of treatment in vestibular neuritis. Although some endorse systemic corticosteroids, there is currently insufficient evidence for routine use.[19] Antiviral medications are ineffective.[20]

#### *Medications*

Initially, vertigo and associated nausea and vomiting can be treated with a combination of antihistamine (dimenhydrinate [Dramamine], 50 mg every 6 hours), antiemetic (promethazine [Phenergan], 25 mg every 6 hours), and benzodiazepine (lorazepam [Ativan], 1–2 mg every 4 hours) medications. Patients with severe symptoms may need to be hospitalized for intravenous fluids and medications. Medications should be discontinued after 2 to 3 days because of their vestibular-suppressant effect. Initial recovery in vestibular neuritis is because of central compensation, followed later by the return of vestibular function. Vestibular suppressants can block central compensation and thus impede resolution if used for prolonged periods.

#### *Vestibular rehabilitation*

As opposed to medications, vestibular exercises hasten recovery in vestibular neuritis. Exercises increase central compensation for the peripheral defect, thereby improving balance, ocular stability, gait, and vertigo. Compared with placebo, vestibular rehabilitation has an odds ratio of 2.67 (95% confidence interval, 1.85–3.86) for symptom improvement.[14] Patients should begin exercises as soon as the acute phase resolves and movement is tolerable, generally within 2 to 3 days of onset. Exercises may be home-based or formally supervised.

## VESTIBULAR MIGRAINE

Vestibular migraine is often an unrecognized cause of episodic vertigo. Patients usually have other migraine symptoms with attacks, such as headache and photophobia, which differentiate it from other causes of vertigo. Treatment is similar to other types of migraine headache.

### *Epidemiology*

Vestibular migraine is a common cause of recurrent, episodic vertigo, but is underrecognized. It is 5 to 10 times more common than Meniere's disease, and approximately 1% of the population is affected at any time.[21,22] Vestibular migraine is more common

among children than adults, with an estimated prevalence of nearly 3% among children aged 6 to 12 years.[23] Among adults, it is three times more common among women than men, and more often seen during the third to fifth decade of life.[21] Family history is a significant risk factor.

### *Pathophysiology*

As a variant of migraine, vestibular migraine has a similar pathophysiology. Although not well-understood, it is theorized that migraine results from central vascular dysregulation and abnormal neuronal activity. Certain triggers may lead to vasoconstriction of the cerebrovasculature and hypoxia, and concurrent neuronal depolarization leads to aura and headache.[24] Other terms for vestibular migraine include migraine-associated vertigo and migrainous vertigo.

### *Clinical Presentation*

As the name suggests, patients with vestibular migraine experience symptoms of vertigo and migraine. Although most patients experience vertigo close in timing with their migraine headaches, the two may not occur simultaneously. Vertigo may precede headache (like a vertiginous aura), coincide with headache, or occur separately. Vertigo can be spontaneous or triggered by position changes. Many patients experience head motion intolerance and visual vertigo (vertigo brought on by watching a moving object, such as traffic). Vertigo may last minutes, hours, or days. If patients do not experience headache with vertigo, then often there are other symptoms of migraine, such as aura, photophobia, or phonophobia. Often, vertigo episodes have similar triggers to migraines, such as lack of sleep, menstruation, and skipped meals. Most patients do not experience aural symptoms, such as hearing loss and tinnitus. Clues distinguishing vestibular migraine from Meniere's disease include an attack lasting days and normal hearing.

The physical examination is normal in patients with vestibular migraine, unless presenting during a vertigo attack. At that time, patients may exhibit signs of imbalance, such as a positive Romberg test.

### *Diagnosis*

Vestibular migraine is a clinical diagnosis and one of exclusion. Diagnostic criteria have been developed that specify definite and probable vestibular migraine (**Box 1**).[25] A "dizzy diary" is helpful to identify symptoms and timing of vertigo in relation to headaches. If the diagnosis remains unclear, vertigo that responds to migraine medications suggests vestibular migraine. There is no confirmatory test for vestibular migraine; however, it is prudent to obtain audiometry and vestibular function tests to exclude other causes. Patients with migraine are more likely than the general population to have vestibular disorders, such as Meniere's disease and BPPV.[26,27]

### *Treatment*

There is little evidence to guide treatment of vestibular migraine, and recommendations are mostly based on expert opinion.[21] However, following principles of migraine management, most patients can attain good control of vertigo.

#### *Lifestyle*

Patients should identify and avoid triggers for their vertigo or migraines. Regular sleep, meals, and exercise benefit most patients.

**Box 1**
**Diagnostic criteria for vestibular migraine**

Definite vestibular migraine

A. Episodic vestibular symptoms of at least moderate severity

B. Current or previous history of migraine according to the 2004 criteria of the International Headache Society (IHS)

C. One of the following migrainous symptoms during two or more attacks of vertigo: migrainous headache, photophobia, phonophobia, visual aura, or other aura

D. Other causes excluded

Probable vestibular migraine

A. Episodic vestibular symptoms of at least moderate severity

B. One of the following: (1) current or previous history of migraine according to the 2004 IHS criteria; (2) migrainous symptoms during vestibular symptoms; (3) migraine precipitants of vertigo in more than 50% of attacks (food triggers, sleep irregularities, or hormonal change); or (4) response to migraine medications in more than 50% of attacks

C. Other causes excluded

***Medications***

For vertigo, vestibular suppressants noted previously, such as dimenhydrinate, promethazine, and meclizine (Antivert, 25 mg every 8 hours) can be helpful. Acute vertigo can be aborted with triptans as used for migraine. Patients with frequent, severe, or prolonged episodes should be offered migraine-prophylactic medications. Preventive medications include β-blockers, anticonvulsants, tricyclic antidepressents, and calcium channel blockers. A reasonable goal is to reduce the frequency of attacks by 50%.

***Vestibular rehabilitation***

For patients with chronic imbalance or vertigo, vestibular exercises may be considered.

## MENIERE'S DISEASE

The triad of episodic vertigo, fluctuating hearing loss, and tinnitus is the hallmark of Meniere's disease. Because of its remitting and relapsing nature, the diagnosis is challenging. However, most patients can be reassured that vertigo can be significantly controlled with lifestyle changes and medication.

### *Epidemiology*

The incidence and prevalence of Meniere's disease is difficult to determine because of its periodic nature. However, its estimated prevalence is 0.2%, with women slightly more affected than men.[28] There is a genetic preponderance of Meniere's; other risk factors include a prior history of vestibular neuritis, head trauma, and syphilic otitis.[29]

### *Pathophysiology*

Endolymphatic hydrops is presumed to be the underlying cause of Meniere's.[30] Dysregulation of fluid results in swelling in the endolymphatic compartment, leading to symptoms of vertigo, hearing loss, tinnitus, and aural pressure. Eventually, swelling may cause permanent damage to vestibular structures. There are multiple potential causes

of endolymphatic hydrops, including genetic, autoimmune, vascular, viral, allergic, and traumatic.[31] Meniere's disease is the term for the idiopathic form. When a cause is identified (prior trauma, syphilic otitis) it is termed Meniere's syndrome.

### *Clinical Presentation*

The classic symptoms of Meniere's disease include vertigo, hearing loss, tinnitus, and aural fullness. Symptoms tend to vary widely among patients. Some patients mainly experience aural symptoms, some mainly vertigo, and others may be equally affected.

Vertigo attacks are acute in onset and severe. Episodes usually last a few hours, but are at least 20 minutes in duration and can last up to 24 hours. Vertigo attacks are often preceded by aural fullness or tinnitus. Vertigo may be described as a spinning or rocking sensation, and may be associated with nausea and vomiting. In some patients, attacks may cluster over a period of a few weeks, whereas others can experience years of remission. Over time, patients may experience positional vertigo or general imbalance between episodes because of progressive loss of vestibular function.

Hearing loss in Meniere's disease is fluctuating (usually in relation to vertigo episodes but may be separate), initially in the low frequencies (**Fig. 4**). As the disease progresses hearing loss becomes permanent and involves all frequencies. Tinnitus is usually described as a roaring sensation, and tends to change pitch and loudness during vertigo attacks. Patients often experience a feeling of fullness or pressure in the involved ear associated with hearing loss.

Otolithic crises of Tumarkin are sudden, random drop attacks that patients with Meniere's may experience. There is no associated loss of consciousness, but their unpredictable nature places patients at risk of serious trauma. Therefore, patients with Tumarkin crises should be aggressively treated.

On physical examination, patients with Meniere's disease may have notable hearing loss and potentially balance or gait difficulty depending on the severity of the disease

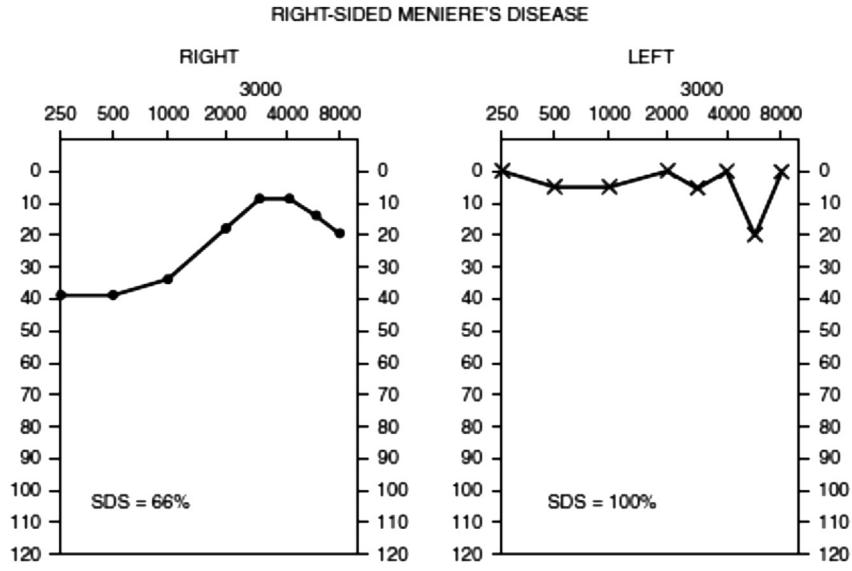


**Fig. 4.** Audiogram of low-frequency hearing loss in Meniere's disease. SDS, speech discrimination score. (*From* Bope ET, Kellerman RD. Conn's Current Therapy 2013. Philadelphia: Saunders; 2012. p. 301; with permission.)

or if presenting during a vertiginous attack. With vertigo episodes, patients manifest a unidirectional, horizontal-torsional nystagmus. Although there may be no physical examination findings in a patient with Meniere's disease, a complete physical examination is crucial to exclude other causes.

### *Diagnosis*

Currently, the diagnosis of Meniere's disease is made clinically because there is no specific diagnostic test for the disease. Diagnostic criteria are listed in **Box 2**. For a definite diagnosis, patients must have had at least two episodes of vertigo lasting at least 20 minutes, audiometrically documented hearing loss on at least one occasion, and tinnitus or aural fullness in the affected ear.[32] Successive audiograms showing fluctuating hearing loss are very helpful, especially if obtained during attacks of vertigo.

#### *Radiographic imaging*

MRI is usually obtained to exclude other causes of vertigo and hearing loss, such as acoustic neuroma, aneurysms, or multiple sclerosis.

#### *Vestibular function testing*

Vestibular testing is not necessary for diagnosis, but may be useful for determining bilaterality of disease and candidates for interventional treatments. Initially, testing may be normal, but with progressive disease most patients show vestibular hypofunction on caloric testing. The vestibular evoked myogenic potential is a newer method used for diagnosis and monitoring, but has not yet been fully validated.[33]

**Box 2**
**Diagnostic criteria for Meniere's disease**

Certain Meniere's disease

Definite Meniere's disease plus histopathologic confirmation

Definite Meniere's disease

A. ≥2 definitive spontaneous episodes of vertigo 20 min or longer

B. Audiometrically documented hearing loss on at least 1 occasion

C. Tinnitus or aural fullness in the treated ear

D. Other causes excluded

Probable Meniere's disease

A. One definitive episode of vertigo

B. Audiometrically documented hearing loss on at least 1 occasion

C. Tinnitus or aural fullness in the treated ear

D. Other causes excluded

Possible Meniere's disease

A. Episodic vertigo without documented hearing loss, or Sensorineural hearing loss fluctuating or fixed, with disequilibrium but non-episodic

B. Other causes excluded

*Adapted from* Committee on hearing and equilibrium guidelines for the diagnosis and evaluation of therapy in Meniere's disease. American Academy of Otolaryngology-Head and Neck Foundation, Inc. Otolaryngol Head Neck Surg 1995;113(3):181–5; with permission.

### *Treatment*

Goals of treatment are to decrease the frequency and severity of vertigo, improve balance, preserve hearing, and improve overall quality of life. Patients should be educated that Meniere's is a chronic condition with no cure, but that with treatment nearly all patients have significant improvement of vertigo. Unfortunately, hearing loss and tinnitus are more difficult to control. Treatment uses a stepwise approach, starting with lifestyle changes and adding diuretics if necessary. Eight percent of patients attain control of vertigo with conservative treatment.[34] If medical management fails, several interventional options are available.

#### *Lifestyle changes*

Many patients can achieve adequate control of symptoms with dietary changes and avoidance of triggers. Episodes can be precipitated by high salt intake, caffeine, alcohol, allergies, nicotine, monosodium glutamate, and stress. Patients should limit salt intake to less than 2 g daily, and limit caffeine and alcohol to one drink daily. Treatment of allergies, including immunotherapy, can significantly improve symptoms.[35]

#### *Medications*

Vestibular-suppressant medications are useful during acute attacks of vertigo. An oral steroid burst is often given as well, although evidence for its efficacy is limited and it is being replaced by intratympanic steroid injection.[36]

If lifestyle changes do not effectively control vertigo, then daily diuretics may be added. A typical regimen is triamterene-hydrochlorothiazide (Dyazide), 37.5 to 25 mg daily, although other diuretics may be used. Betahistine (Serc) is a vasodilator and antihistamine commonly used in Europe for prophylaxis, but it is not available within the United States.[37]

#### *Vestibular rehabilitation*

Vestibular exercises may be beneficial for patients who experience imbalance or disequilibrium between attacks of vertigo, and for the elderly to reduce falls.[14]

#### *Hearing aids*

Patients with bilateral hearing loss can benefit from hearing aids. However, because of the fluctuating nature of hearing loss, many patients become frustrated with hearing aids.

#### *Intratympanic glucocorticoids*

Injection of glucocorticoid into the middle ear through the tympanic membrane can improve vertigo in many patients. Although less effective than other treatments, it is minimally invasive and has the potential to improve hearing.[36] Approximately 40% of patients achieve complete control of vertigo with intratympanic glucocorticoid injections.[38,39] One trial conducted over 2 years found that 90% of patients had adequate control of vertigo with repeated steroid injection, obviating the need for more invasive treatment.[40]

#### *Meniett device*

The Meniett device is a portable machine that delivers pulses of positive pressure to the middle ear through a tympanostomy tube. Theoretically, this controls symptoms by improving endolymphatic drainage. Although there is limited evidence that the Meniett device is effective, it only requires placement of a tympanostomy tube and does not cause hearing loss.[41]

***Endolymphatic sac procedures***
Endolymphatic sac procedures involve drainage of endolymphatic fluid by placing a shunt to the mastoid and/or decompression. There is some controversy regarding the effectiveness of the procedures, but there is minimal risk of hearing loss and reports of long-term effectiveness in 75% of patients.[15,42] This procedure is generally indicated for patients who have failed prior medical treatment and wish to preserve hearing.

***Intratympanic gentamycin***
Gentamycin is an aminoglycoside antibiotic that is vestibulotoxic and cochleotoxic. Intratympanic gentamycin causes a "chemical labyrinthectomy" when injected through the middle ear to be absorbed by the inner ear. Studies report more than 95% effectiveness; however, up to one-third of patients have permanent hearing loss.[43,44] Therefore, it is often reserved for treatment of patients with intractable vertigo and significant hearing loss.

***Vestibular neurectomy***
Severing the vestibular nerve is the most definitive treatment for controlling vertigo when the goal is to preserve hearing, achieving control in 95% of patients.[45] However, neurectomy requires general anesthesia, a craniotomy, and overnight monitoring in an intensive care unit. It also has several potential risks, such as facial nerve damage.

***Labyrinthectomy***
Surgical destruction of the labyrinth is a highly effective method for eliminating vertigo, but also causes irreversible hearing loss. It is therefore reserved for patients with intractable vertigo who have failed other treatments and have no serviceable hearing in the involved ear.[31]

***Bilateral disease***
Bilateral Meniere's disease affects approximately 15% of patients. However, over time it is estimated that up to 30% or more develop disease in both ears.[46] Bilateral Meniere's disease presents a treatment challenge. Treating one side may not relieve vertigo, and interventional treatment can cause permanent hearing loss in a patient already at risk of hearing loss in both ears.

## SUMMARY

Vertigo is a common problem that often brings patients into the primary care office. BPPV, vestibular neuritis, vestibular migraine, and Meniere's disease are the four most common causes of vertigo. Because these conditions are mainly diagnosed based on clinical grounds, clinicians should be familiar with their history, physical examination findings, and diagnostic criteria. With this armament, primary care physicians should feel well-equipped to aid the dizzy patient.

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